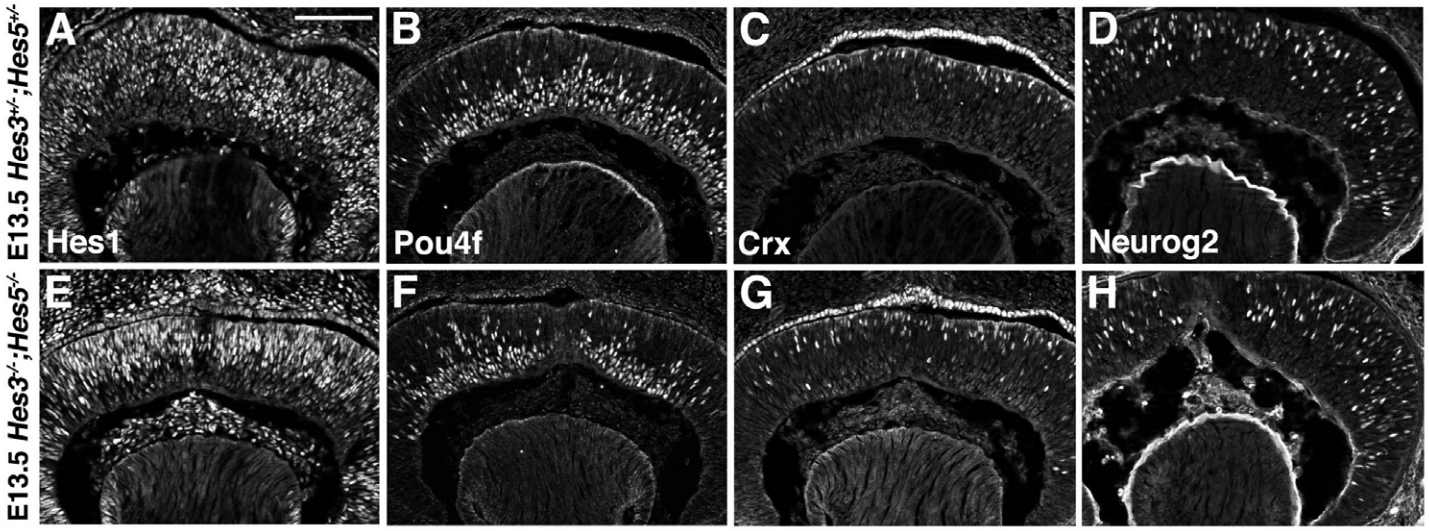
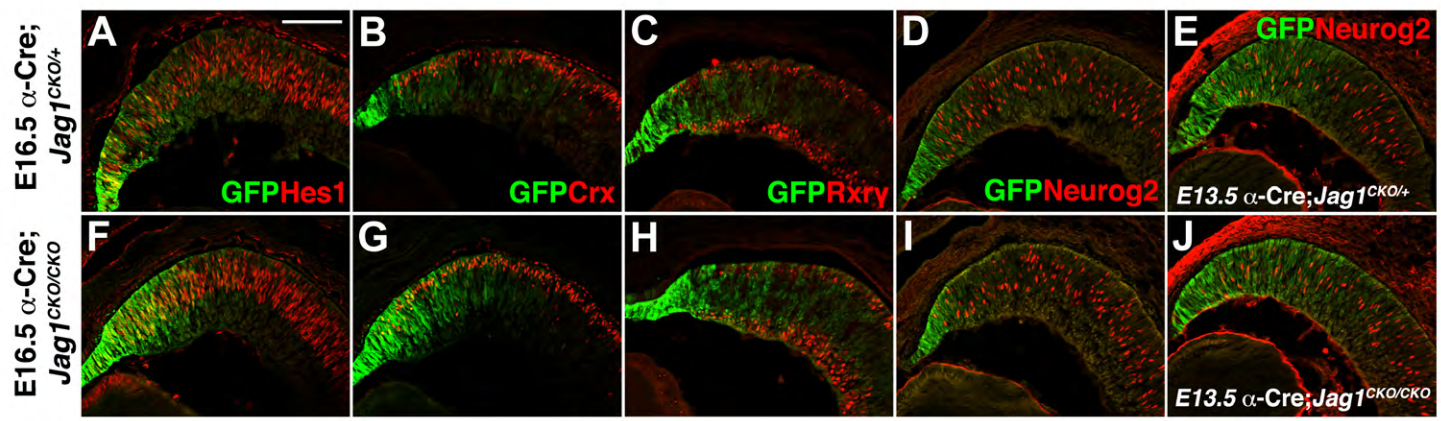
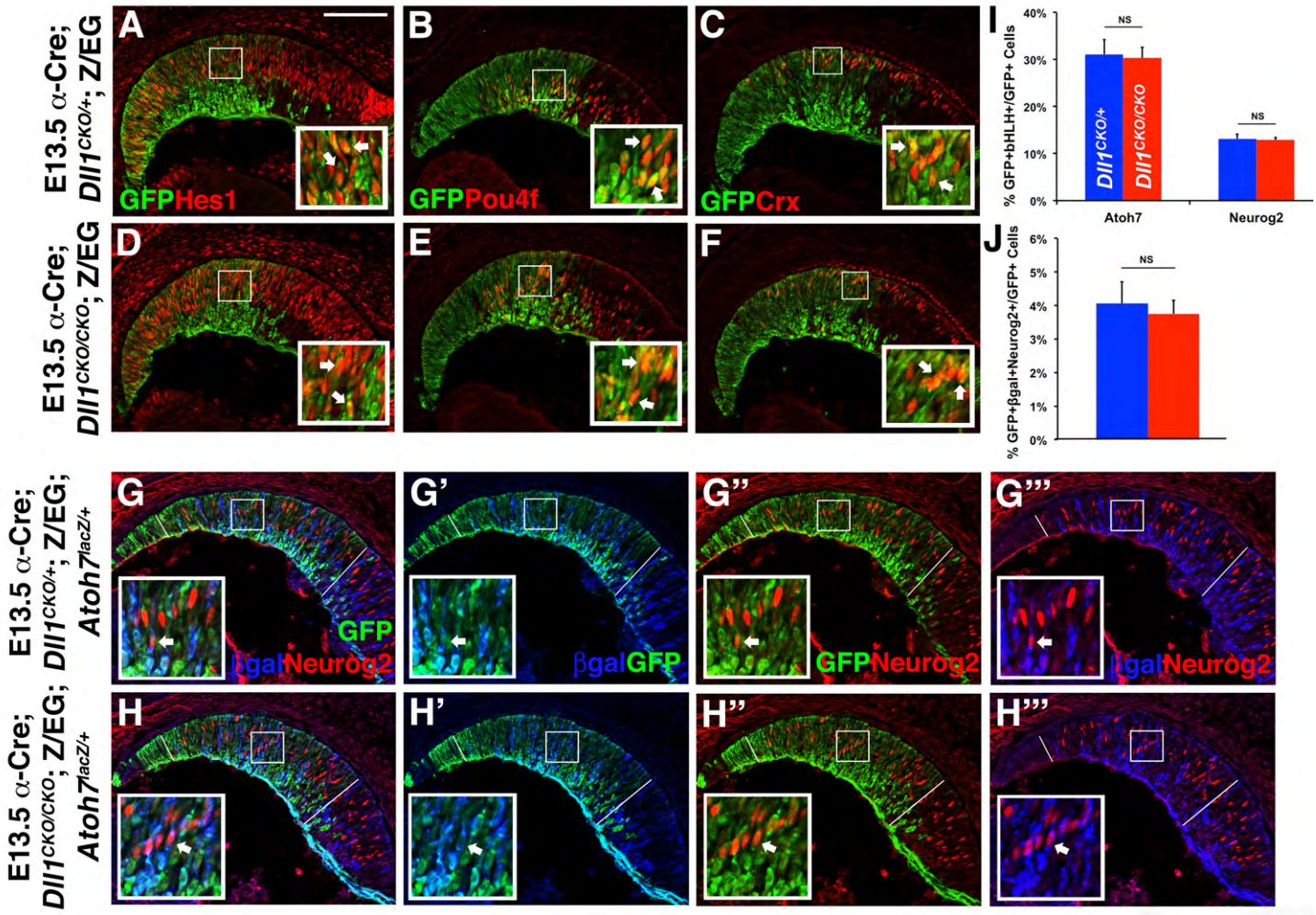


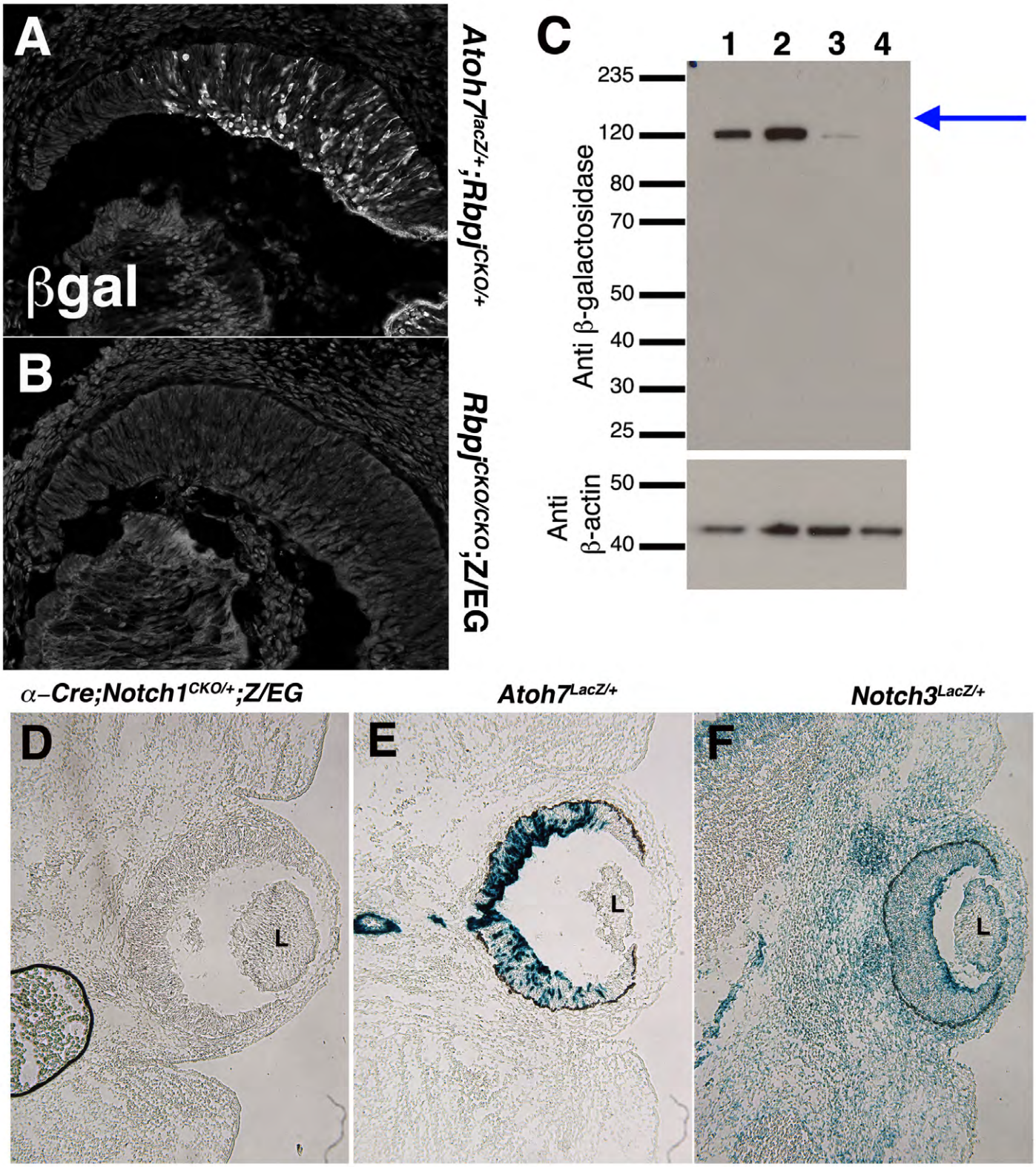
Supplemental Figure 1. *Hes3* and *Hes5* are not required for Neurog2 expression. *Hes3*$^{-/-}$;*Hes5*$^{-/-}$ mutants have no obvious changes in Hes1+ RPCs (**A, E**), Pou4f+ RGCs (**B, F**), Crx+ photoreceptor precursors (**C, G**) or Neurog2+ RPCs (**D, H**). Scale bar in A =100µm; n≥3 embryos per genotype.



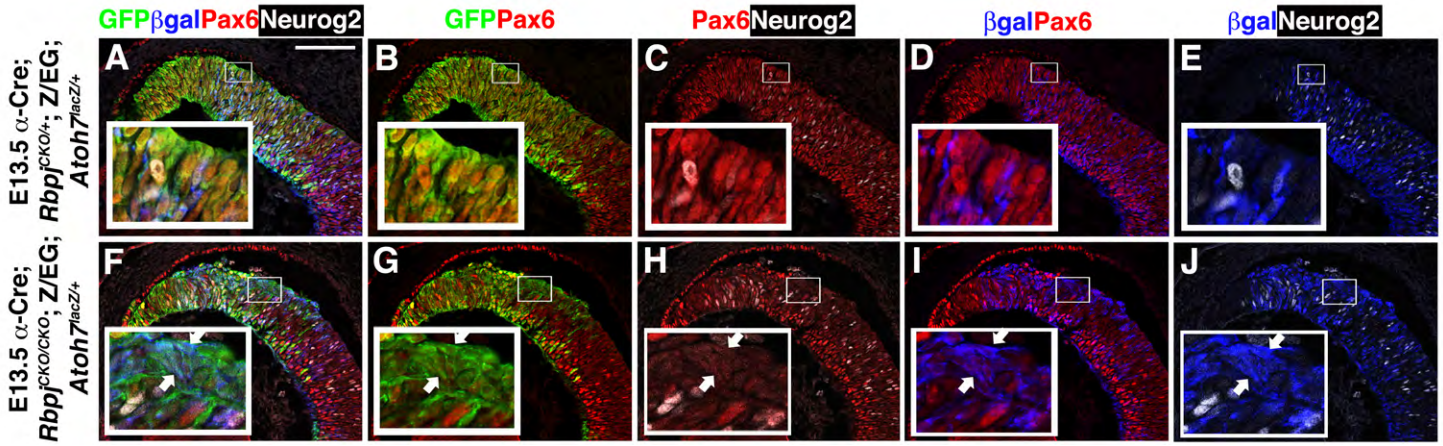
Supplemental Figure 2. Loss of *Jag1* in the distal optic cup has no effect on retinal neurogenesis. A-D, F-I) E16.5 α-Cre;*Jag1*$^{CKO/CKO}$ mutant retinas have no discernable phenotype when compared to α-Cre; *Jag1*$^{CKO/+}$control retinas. The distribution of Hes1+ RPCs (**A, F**), Crx+ photoreceptor precursors (**B, G**), Rxrg+ early cones or RGCs (**C, H**) appears normal after conditional removal of *Jag1.* Neurog2+ cells were also unaffected at E13.5 (**E, J**) and E16.5 (**D, I**). Scale bar in A =100µm; n≥3 embryos per genotype.



Supplemental Figure 3. *Dll1* is not required for *Atoh7*^{lacZ} or *Neurog2* retinal expression. A-F) Loss of *Dll1* from the distal retina resulted in fewer Hes1+ RPCs (**A, D**), but increased Pou4f+ RGCs (**B, E**). Crx+ photoreceptor precursors were unaffected in α-Cre;*Dll1*$^{CKO/CKO}$;Z/EG mutants, compared to controls (**C, F**). **G-G'''**) Highly overlapping expression of *Atoh7*^{LacZ} (β-gal) and Neurog2 in α-Cre;*Dll1*$^{CKO/+}$; Z/EG;*Atoh7*$^{lacZ/+}$ retinas. **H-J**) Loss of *Dll1* did not perturb the β-gal+, Neurog2+, or β-gal+Neurog2+ cohorts, cells between the white lines were quantified in **I** and **J**. Arrows within each inset point to α-Cre lineage cells (GFP+) coexpressing each marker. Scale bar = 100μm; boxed areas at high magnification within insets. NS = not significant, n≥3 embryos per genotype, with error bars indicating SEM.



Supplemental Figure 4. A subcolony of Z/EG transgenic mice lacking constitutive β-geo activity. A, B) *Atoh7*$^{lacZ/+}$;*Rbpj*$^{CKO/+}$ retinal sections have robust β-gal staining (demarcating the *Atoh7* lineage); whereas, constitutive β-geo expression is absent from *Rbpj*$^{CKO/CKO}$;Z/EG eyes. **C)** Western blot of E13.5 eye total protein: Lane 1 = *Atoh7*$^{LacZ/+}$, Lane 2 = *Atoh7*$^{LacZ/+}$;Z/EG Tg/+, Lane 3 = Z/EG Tg/+, Lane 4 = normal adult brain. Lanes 1 and 2 have a single band matching the size of the β-galactosidase reporter expressed from the *Atoh7* locus (118.8 kDa). However, there no detectable beta-geo fusion protein (137 kDa, blue arrow at right), from the Z/EG transgene, although faint β-galactosidase protein is present in Lane 3. **D-F)** E13.5 cryosections incubated together for 48 hours at 37°C in x-gal chromogenic staining solution. There is no β-galactosidase activity throughout the head of Z/EG embryos, whereas all cells are predicted to be x-gal+ **(D)**. X-gal+ cells are evident in the correct patterns in **E** and **F**.



Supplemental Figure 5. *Pax6* is required for *Neurog2* expression, but not *Atoh7,* in Zone 2 retinal cells. A-E) In control retinas, Pax6 coexpresses with both Neurog2 and *Atoh7*^{lacZ} (β-gal+). **F-J)** In the absence of *Rbpj,* Pax6 expression is reduced in zone 2 retinal cells, which also lack Neurog2 (**F-H, J**). However, *Atoh7*^{lacZ} expression is maintained within this population (**I-J**). Scale bars = 100μm; boxed areas at higher magnification in insets. Arrows within each inset point to α-Cre lineage cells (GFP+) that are Pax6-neg, Neurog2-neg, and *Atoh7*^{lacZ}+. n≥3 embryos per genotype.